Nematology Circular No. 26
March-April 1977

Fla. Dept. of Agric. & Consumer Serv.
Division of Plant Industry

THE ROLE OF NEMATODES IN DISEASE COMPLEXES

J. G. Baldwin

Introduction

A diseased plant affected by only a single pathogen is rare. Nematodes, acting alone, can sap the vitality of a plant, but they can also facilitate infection by additional pathogens. Such combinations of nematodes with fungi, bacteria, or viruses may act synergistically to incite disease; that is, the interacting effect of 2 or more disease agents may be greater that the additive effect of the pathogens acting independently. Ways in which nematodes participate in disease complexes include the following: serving as vectors or agents of pathogen transmission, providing portals of entry, inducing necrotic infection courts, modifying the physiology of the host, of "breaking" mechanisms of resistance to other pathogens.

The Nature of Disease Complexes

Transmission of disease agents: Many viruses need a biological agent for inoculation and successful transmission to a suitable host. Although most vectors are arthropods, a few nematode genera, Longidorus (needle nematode), Xiphinema (dagger nematode), and Trichodorus (stubby-root nematode), transmit certain "soil-borne" viruses (20). Multiplication of viruses within a nematode vector has not been detected, but specificity of virus-nematode relationships suggests that more than simple mechanical transmission and wounding of the host is involved (17).

Nematodes also transmit certain fungi and bacteria which can incite diseases. For example, the fungus, Dilophospora alopecuri Fries is introduced into the apical meristem of wheat by Anguina tritici (Steinbuch) Chitwood (wheat gall nematode). Attempts to produce the disease in the absence of the nematode apparently have been unsuccessful (1,9). Similarly, Ditylenchus dipsaci (Kühn) Filipjev (stem nematode) sometimes transmits the casual agent of bacterial wilt of alfalfa, Corynebacterium insidiosum (McCulloch) Jensen, and feeding by the nematode results in greater wilt severity than when the bacterium occurs alone (6).

Wounding: Plant-parasitic nematodes wound host plants, and wounds provide ports of entry for other pathogens. Pitcher (17) noted that such wounds apparently favor bacteria more than fungi, because bacteria are less well adapted for penetrating the host's epidermis. Disease symptoms similar to those which occur in nematode-bacterial wilt interactions were simulated by substituting mechanical injury for nematode feeding (10,12). On the other hand, mechanical injury did not appear to be an adequate substitute for the role of the nematode in nematode-fungal wilt interactions (8,22).

Necrosis: Wounding of a host, by some nematodes, results in decay of root tissues, which may favor ingress of certain additional pathogens. These pathogens are often unspecialized and may be facultative parasites; that is, they generally survive on dead plant tissue, but are also capable of invading living tissue. For example, the fungus, Fusarium oxysporum f. vasinfectum (Atkinson) Snyder and Hansen grew well in decaying cortical tissue associated with Meloidogyne incognita (Kofoid and White) Chitwood (root-knot nematode) on cotton, but did poorly in healthy tissue (13). Furthermore, fungal species of Curvularia, Botrytis, Aspergillus, and Penicillium, which do not normally parasitize tobacco, may be pathogenic when root-knot nematodes are present (19). Lesions produced by Radopholus similus (Cobb) Thorne (burrowing nematode) may become foci for root-rot complexes on banana (11). DuCharme (3) has suggested that similar complexes may be involved in spreading decline of citrus. Similarly, Pratylenchus penetrans (Cobb) Filijev and Stekhovan (lesion nematode) aids root rot of peach (15) and strawberry (14).

Physiologically modified substrate: Creation of an infection court is one way in which nematodes modify a host to enhance infection by additional pathogens. However, there is increasing evidence that nematodes modify host substrates in more subtle ways. Changes in biochemistry of the host are probably the most important factors favoring disease complexes involving nematodes (23). Nematodes may induce production of host metabolites which are favorable to other pathogens, or they may destroy host metabolites that provide resistance to potential pathogens (17). Meloidogyne sp. induces gross physiological changes in a host. Thus, infection with root-knot nematodes prior to inoculation with a bacterial or fungal pathogen is more likely to result in a synergistic disease complex, than when inoculations are simultaneous. Apparently, the nematodes substantially alter host physiology so that a subsequently introduced pathogen is favored (19, 24). Synergistic interactions on a host may occur, even when the pathogens are isolated on different halves of a root system; this suggests that some nematode systemically alter host physiology (4).

Physiological changes induced by root-knot nematodes may also result in complexes with viruses. Soybeans infected by root-knot nematodes and the tobacco ring-spot virus had extensively galled and stunted root systems. The nematode alone resulted in galling, but did not reduce root size; tobacco ring-spot virus alone did not noticeably affect soybean roots (21). On the other hand, tobacco ring-spot virus on tobacco has been noted to substantially increase host penetration by Meloidogyne javanica (Treub) Chitwood (2)

Nematodes other than Meloidogyne spp. may produce physiological changes in a host, which promote disease complexes. For example, digestive enzymes recovered from D. dipsaci and injected into onion bulbs, resulted in much more severe infection by Botrytis alli Munn than occurred in bulbs injected only with water (16). Such alteration in host physiology by nematodes is thought to be the most important factor in disease complexes which include nematodes.

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"Breaking" disease resistance: Nematodes may alter hosts to such an extent that these hosts may become susceptible to organisms to which they are otherwise resistant. Root-knot nematodes enable races of Fusarium to attack tomatoes ordinarily resistant to the fungus race (5,25), and Fusarium-resistant tobacco may become highly susceptible if root-knot nematodes are present (18). Alfalfa cultivars with high resistance to wilt by C. insidiosum may be diseased by this bacterium when D. dipsaci is present (7).

Conclusions

Physiological variation in a given nematode species may be partially responsible for inconsistency in susceptibility of a given host species. Disease complexes may also explain why a nematode species is in some cases associated with much more host damage than the same nematode in other similar situations. Furthermore, it has become increasingly apparent that parasitic nematodes may be involved in disease complexes, not simply by providing wounds or ports of entry for other organisms, but often by little understood biochemical and physiological alterations of the host plant. Powell (19) has observed that disease complexes may be the major economic hazard posed by nematodes. Certainly an improved understanding of these relationships involving nematodes will enhance our ability to control plant diseases.

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